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Recommended Citation

Nicholas J. Kaplinsky. (2009). "Temperature Compensation Of Auxin Dependent Developmental Patterning". *Plant Signaling And Behavior*. Volume 4, Issue 12. 1157-1158.
<https://works.swarthmore.edu/fac-biology/41>

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Temperature compensation of auxin dependent developmental patterning

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Key words: *BOBBER1*, *BOB1*, temperature, development, homeostasis, heat shock granule, sHSP

Submitted: 08/27/09

Accepted: 08/27/09

Previously published online:

www.landesbioscience.com/journals/psb/article/9949

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Addendum to: Perez DE, Hoyer JS, Johnson AI, Moody ZR, Lopez J, Kaplinsky NJ. *BOBBER1* is a non-canonical Arabidopsis small heat shock protein required for both development and thermotolerance. *Plant Physiol* 2009; 151:241-252; PMID: 19571304; DOI:10.1104/pp.109.142125. and Jurkuta RJ, Kaplinsky NJ, Spindel JE, Barton MK. Partitioning the apical domain of the Arabidopsis embryo requires the *BOBBER1* NudC domain protein. *Plant Cell* 2009; 21:1957-71; PMID: 19648297; DOI: 10.1105/tpc.108.065284.

The establishment of localized auxin gradients plays a central role in developmental patterning in plants. Auxin levels and responses have been shown to increase with temperature although developmental patterning is not affected. This suggests the existence of a homeostatic mechanism that ensures that patterning occurs normally over a range of temperatures. We recently described the cloning and characterization of *BOBBER1* (*BOB1*), an Arabidopsis gene which encodes a small heat shock protein. *BOB1* is required for the establishment of auxin gradients and for normal developmental patterning. *BOB1* is also required for organismal thermotolerance and localizes to heat shock granules at elevated temperatures. Since *BOB1* functions in both temperature responses and developmental patterning we propose that *BOB1* may encode a component of a developmental temperature compensation mechanism.

Polar auxin transport and the resulting establishment of local auxin gradients is a central theme in plant developmental patterning. Auxin acts as a morphogen which establishes patterning and cell identity in the female gametophyte and is subsequently required for the establishment of polarity and patterning during embryogenesis.^{1,2} During vegetative plant development the correct establishment of auxin gradients is required for the establishment of phyllotaxy and for sculpting the shape of leaf margins among many other patterning functions.³ These patterning events occur in a stereotypical manner regardless of changes in temperature.

Given that these important developmental functions proceed normally over a range of temperatures it is perhaps surprising that auxin levels and responses are significantly influenced by changing temperatures. Gray et al. showed that there is a ~1.75x increase in free IAA levels when Arabidopsis plants are grown at 29°C instead of at 20°C.⁴ Increased IAA levels at elevated temperatures are accompanied by auxin dependent hypocotyl elongation as well as changes in the patterns of auxin responsive gene expression. However, changes in developmental patterning were not reported. This suggests the existence of a robust homeostatic mechanism which normalizes auxin mediated developmental patterning events in response to changing temperatures.

We recently characterized *BOBBER1* (*BOB1*) which encodes a non-canonical small heat shock protein (sHSP) with functions both in developmental patterning and in high temperature responses.^{5,6} *BOB1* is an essential gene and *bob1-1* null mutants arrest as globular embryos. In *bob1-1* mutants a DR5:GFP reporter was used to show that there is a failure to establish auxin gradients during embryogenesis with resulting patterning abnormalities. A similar lack of auxin gradients is observed in embryos treated with 2,4-D, which, like *bob1-1* embryos, arrest at the globular stage.⁷ In the apical half of *bob1-1* embryos the central meristematic domain is expanded to encompass the lateral domains where cotyledons would normally form. Similarly, in the basal half of *bob1-1* embryos auxin maxima are not established and the expression of *SCARECROW* (*SCR*) at the root pole is

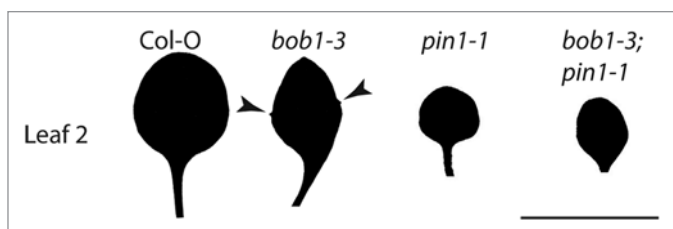


Figure 1. Leaf morphology of Col-O, *bob1-3*, *pin1-1* and *bob1-3; pin1-1* plants. The margins of leaf 2 of Col-O plants but are completely smooth, while serrations are present on the margins of *bob1-3* leaves (arrow heads). No serrations are present on *pin1-1* leaves, and *pin1-1* is epistatic to *bob1-3* in *bob1-3; pin1-1* double mutants. Scale bar is 1 cm.

never observed. This observation is consistent with a requirement for a localized auxin maxima in the formation of the root meristem.⁸

During post-embryonic development *BOB1* is required for normal growth as well as for leaf and floral patterning. When *bob1* activity is genetically reduced pin-formed inflorescence meristems are observed. Pin-formed meristems reflect a lack of organ primordium initiation. This phenotype is specifically observed in mutants including *pinoid* (*pid*), *pin-formed1* (*pin1*) and *yucca1,4,6* triple mutants in which the ability to establish auxin maxima has been disrupted.⁹⁻¹¹ While the presence of phenotypes that phenocopy auxin deficiencies is not evidence that *BOB1* directly modulates auxin mediated patterning, these observations are certainly suggestive. Given the extensive overlap between *bob1* phenotypes and auxin phenotypes a plausible hypothesis is that *BOB1* may be required for normal auxin transport or signal transduction.

bob1-3 is a partial loss of function allele which has, among other phenotypes, serrated leaf margins on early leaves.⁶ Normal (Col-O) plants do not produce serrations on the margins of their first two leaves, however *bob1-3* mutant plants have one or two leaf serrations on leaves one and two. The formation of leaf margin serrations has been shown to require the activity of the auxin efflux carrier *PIN1*.¹² In order to investigate whether the leaf margin phenotype observed in *bob1-3* mutants requires normal *PIN1* activity we crossed *bob1-3* plants to *pin1-1* mutant plants to

generate *bob1-3; pin1-1* double mutants. The margins of the first leaves of double mutant plants are smooth. This demonstrates that the serrated leaf phenotype of *bob1-3* mutants depends on, and modulates, normal *PIN1* activity (Fig. 1).

In addition to characterizing *BOB1*'s developmental functions we also demonstrated that *BOB1* encodes a small heat shock protein (sHSP) with protein chaperone activity. *BOB1* protein can prevent the thermal denaturation of a model substrate in vitro and co-localizes to heat shock granules with canonical sHSPs. *bob1-3* mutants also have thermotolerance defects.⁶ sHSP chaperones function by binding to partially unfolded substrates and preventing their irreversible aggregation.¹³ Assuming that *bob1* developmental defects are due to a change in chaperone activity this would suggest a model where *BOB1* modulates the function of developmental pathways at normal temperatures by ensuring the correct folding of target proteins. One possible target of *BOB1* activity could be signal transduction molecules, perhaps in the auxin signaling pathway, which require stabilization in the absence of ligands or interaction partners, similar to Hsp90 targets.¹⁴

Developmental homeostasis of auxin mediated events over a range of temperatures requires a mechanism which responds to temperature and also modulates auxin responses. We propose that *BOB1* might be a component of this homeostatic mechanism as it has both of these characteristics. sHSPs like *BOB1* are known to undergo temperature dependent

conformational changes that result in altered substrate interactions.¹³ In addition to a role in temperature responses we have shown that *BOB1* is required for the establishment of auxin gradients and appears to modulate auxin mediated developmental events throughout the plant life cycle. We are currently investigating this hypothesis in order to obtain a better understanding of how plants develop normally over a wide range of temperatures.

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